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Strategy for the Treatment of Acute Thromboembolic Stroke Involving an Internal Carotid Artery

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Summary

With the recent improvement of endovascular techniques, intra-arterial local fibrinolytic therapy has become widely available for treatment of acute embolic stroke and there is some evidence that it could be superior to conventional approaches[1-6].

However, because of high mortality and morbidity, strokes involving the internal carotid artery (ICA) and featuring acute thromboembolic occlusion remain problematic[7].

We have successfully performed intra-arterial local fibrinolytic therapy via the anterior communicating artery through the contra-lateral ICA in two consecutive cases of thromboembolic occlusion of the ICA, anterior cerebral artery (ACA) and middle cerebral artery (MCA), and obtained satisfactory results. We here present details of this new technique applied for the two cases and discuss the efficacy of this method compared with conventional approaches.

Introduction

Intra-arterial local fibrinolytic therapy for strokes involving the internal carotid artery caused by acute thromboembolic occlusion frequently lead to enlargement of cerebral infarction because of inadequate re-canalization with the long time taken for thrombolysis and to haemorrhagic complications due to excessive doing with fibrinolytic agents[7].

In order to decrease the time for endovascular surgery and the required dose, we have developed a new technique to recover the blood flow of the middle cerebral artery via the anterior communicating artery through the intact contra-lateral ICA, differing in major aspects from conventional intra-arterial local fibrinolytic therapy.

Material and Methods

Our criteria for thrombolytic therapy are in accordance with those of the PROACT study[8]. In the present two cases, conventional angiography and local intra-arterial fibrinolytic therapy were performed with a transfemoral approach under local anesthesia. Heparinizaion was conducted by delivery of 2,000 units by one-shot injection then continued during endovascular surgery. ACT was controlled between two and three times.

We conducted by advancing a micro-catheter from the contra-lateral internal carotid artery into the anterior communicating artery and then through the A1 on the affected site in the reverse direction to the middle cerebral artery (figure 1).

Micro-ferret catheters (Cook Ltd. Co.) with Tansend-14-standard guide wire (Boston-scien-

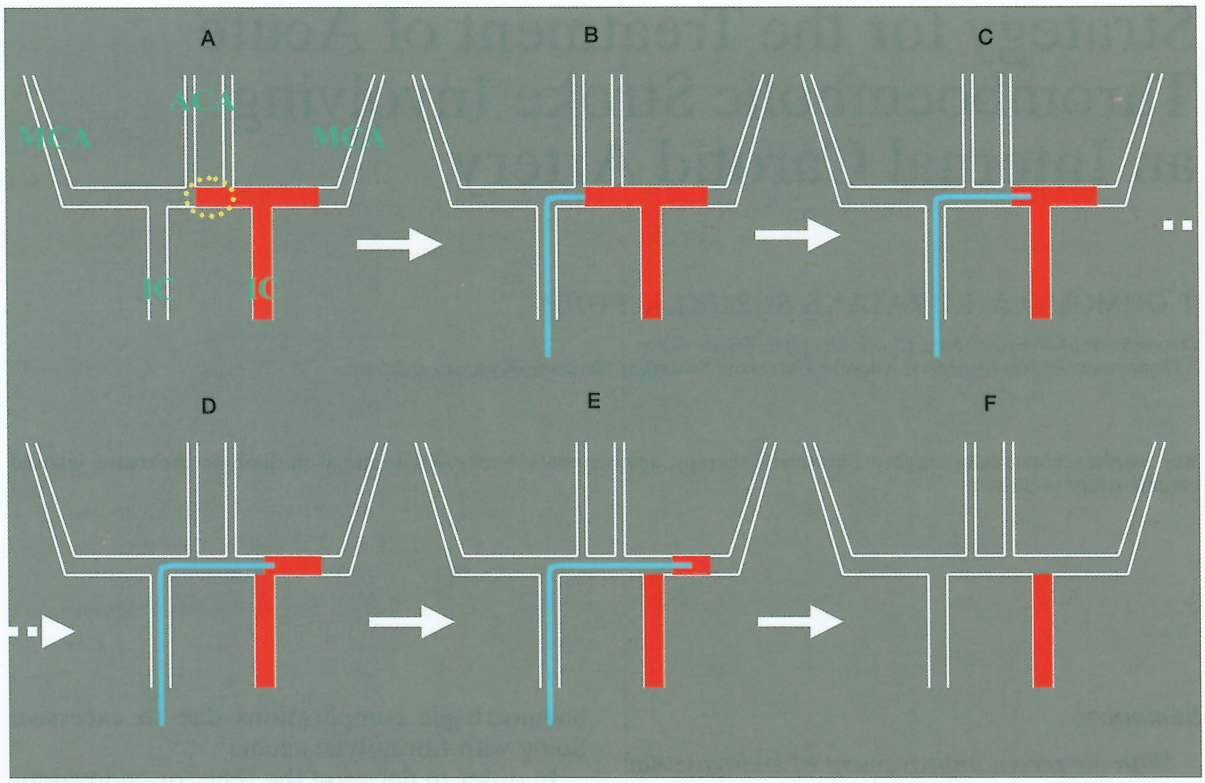

Figure 1 This illustrations shows flow chart of a trans anterior communicating arterial local fibrinolytic theraphy. White lines are vessels of anterior circulation, yellow circle is anterior communicating artery, red area is thromboembolus, blue line is micro-catheter. A) is initial view, F) is final view. B) A micro-catheter was introduced to the point of A1, A2 junction on the side of non occlusion under preceding a micro-guide wire and 120,000 units of the agent was continuously injected. C) A micro-catheter was let into the point of A1 through anterior communicating artery and the 60,000 units were introduced. D) A micro-catheter was let into the point of IC-tip through A1 and the 60,000 units were continuously introduced. E) This procedure was continuously performed to M1 point on the side of the occlusion. F) Blood flow was recovered at the total dosage of 600,000 units.

tific Ltd, Co.) and Agility-10-soft-tip wire (Cordis, Jhonson & Jhonson, Co.) were used. As the thrombolytic agent, 60,000 units of Urokinase were applied in 10 ml of saline continuously infused at the rate of 100 ml/hr. In the results both patients received the same amount but over different periods of time.

The micro-catheter was first introduced to the point of the A1, A2 junction and 120,000 units of the agent were continuously infused. Consequently A2 on the side of the occlusion could be visualized through the anterior communicating artery.

The micro-catheter was slowly advanced to the point of A1 in the same way and a further 60,000 units were introduced. This procedure was repeated until the M1 point on the side of the occlusion. Blood flow was recovered with a total dose of 600,000 units.

Representative Case

Case 1

A 70-year-old male had a history of atrial fibrillation, but was not under medication. After waking at 6:00 as normal he suffered sudden onset of right hemiplegia and aphasia 30 minutes later. He was transferred to our hospital by an ambulance within 35 minutes after onset and CT was performed 1hour after onset. Early CT signs were not present (figure 2) and wide ischemia was not demonstrable from MRI diffusion images (figure 3 upper panel). MRI T2 weighted images showed disappearance of flow voids in the left A1 and M1 (figure 3 lower panel). MRA allowed the left A1 and M1 to be slightly visualized, which suggested the presence of collateral circulation through the anterior communicating artery (figure 4). Those ex-

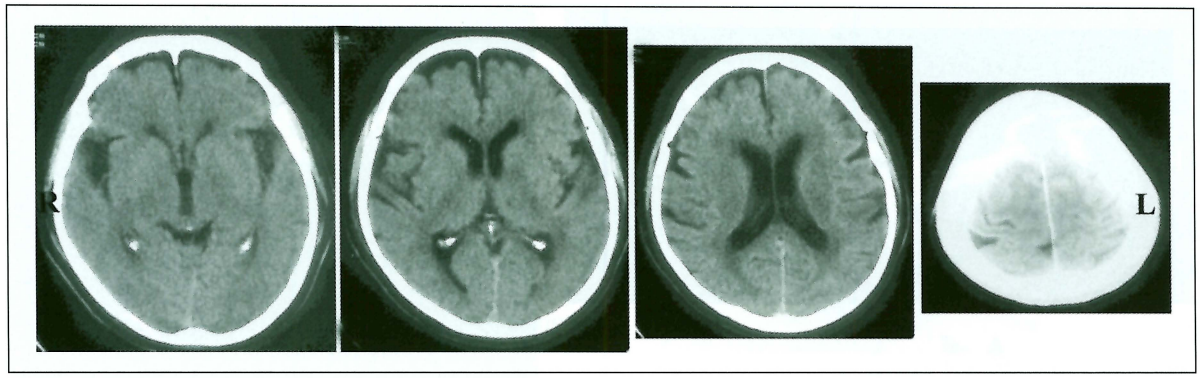

Figure 2 Initial CT image, axial view, showing no early CT signs.

aminations were completed in advance of angiography, performed 2.5 hours after onset. Left carotid angiography revealed complete occlusion of the left internal carotid artery at just after the carotid bifurcation (figure 5: left). The anterior communicating artery and Left anterior and middle cerebral arteries were not demonstrated on right internal carotid angiography (figure 5: center). Almost all of the left middle cerebral arteries were not delineated on vertebral angiography (figure 5: right).

Local intra-arterial fibrinolytic therapy was started 3 hours after onset and accomplished within five hours with a total urokinase dose of 600,000 units. At the end of the procedure, the left anterior and middle cerebral arteries could be demonstrated through the anterior communicating artery on right internal carotid angiography (figure 6).

From the day after these procedures, dosage of 200 mg of Cyclovisine per day was initiated, together with rehabilitation measures. Recovery from the initial neurological deficit was obvious and the right hemiplegia was improved to 4/5 MMT and he could walk without help. Aphasia was also improved to the extent that the patient

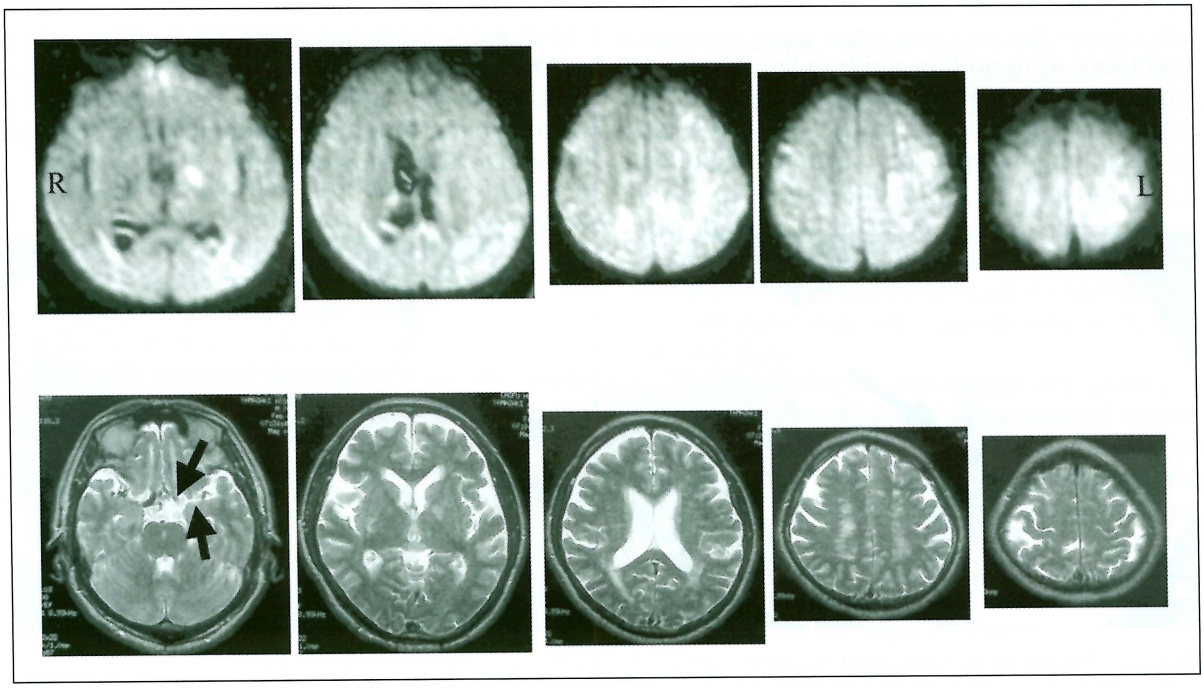

Figure 3 Initial MRI image, axial view, diffusion image (upper) and T2-weighted image showing no vide ischemia and absence flow void of left A1 and M1 proximal (arrow).

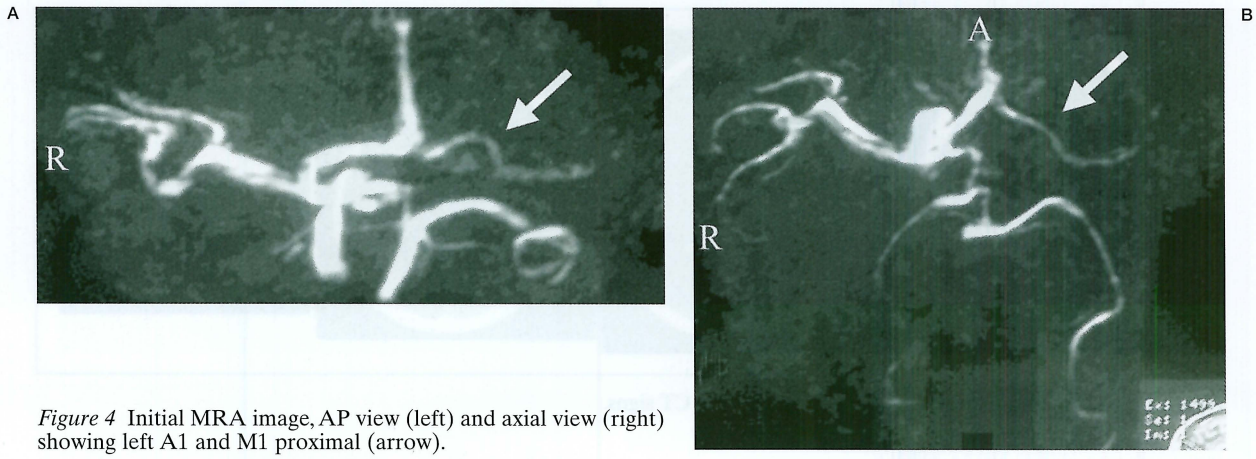

Figure 4 Initial MRA image, AP view (left) and axial view (right) showing left A1 and M1 proximal (arrow).

had no difficulty in activity of daily life. Follow-up MRI two months after discharge showed that the cerebral infarction was localized in the territory of a peripheral middle cerebral artery.

Case 2

A 46-year old male suffering form uncontrolled atrial fibrillation and hyperlipidemia fell down suddenly while in his working place. The patient was transferred to our hospital by ambulance 30 minutes after onset and right hemiplegia and aphasia were present on admission. A CT examination was performed 40 minutes after onset but no early signs were recognized and there were no flow voids in left A1 and M1 observed with MRI T2 weighted imaging. The left A1 was slightly visualized by MRA, suggesting the presence of collateral circulation through the anterior communicating artery. Serial examinations were completed in advance of angiography, performed 2.5 hours after onset. Left carotid angiography revealed complete occlusion of the left internal carotid artery in the cavernous portion. The anterior communicating artery, and left ACA and left MCA were not visualized on right internal carotid angiography. Almost none of the left middle cerebral arteries could be visualized on vertebral angiography, as in case 1. Local intra-arterial fibrinolytic therapy was started two hours 50 minutes after onset and was accomplished within three hours and 10 minutes by means of a total urokinase dose of 600,000. The left anterior and middle cerebral arteries could then be demonstrated through the anterior communicating on right internal carotid angiography.

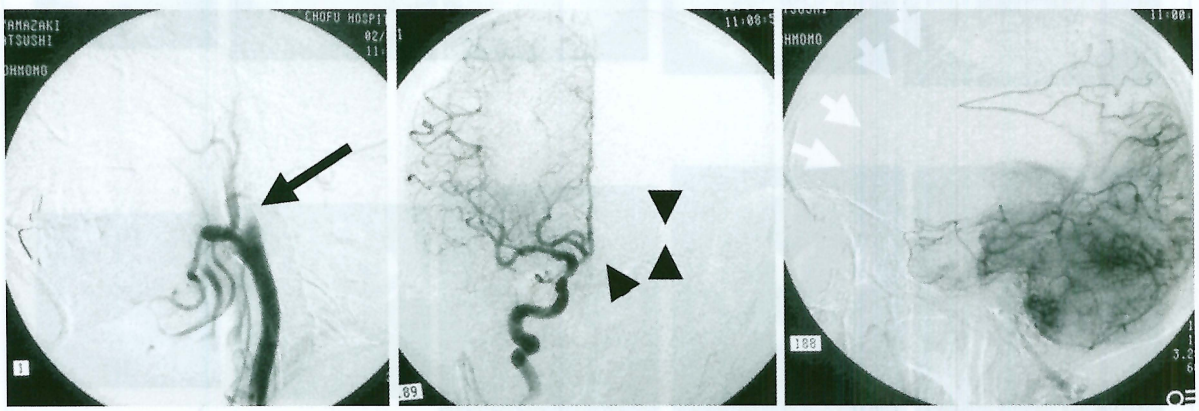

Figure 5 Angiogram A) Left common carotid angiography, lateral view, showing complete occlusion of the left internal carotid artery at just after external-internal bifurcation (black arrow) B) Right internal carotid angiography, AP view, showing no filling to anterior commucating artery, left A1 and M1 (black arrow head). C) Left verterography, lateral view, showing slightly filling to middle cerebral territory (white arrow).

From the day after these procedures, dosage with 200 mg of Cyclovisine per day and rehabilitation were started. Left hemiplegia was improved to 4/5 MMT for the lower but not the upper limb and he could walk by himself Motor aphasia remained with slight improvement, but sensory aphasia was greatly improved so that the patient become capable of independent activity of daily life.

In comparison with Case 1, the cerebral infraction of case 2 involved a wide area, encompassing both perforator and peripheral sections of the middle cerebral artery.

Discussion

Administration of fibrinolytic agents for cerebral infarction has been achieved via both trans-venous [3,4] and trans-arterial routes [2]. Recent developments in endovascular techniques now allow intra-arterial local fibrinolytic therapy [1,7]. This has advantages as detailed below, although as an invasive procedure requiring longer periods of time to start compared with intravenous fibrinolytic therapy it is not without problems. Most importantly, however, intra-arterial local fibrinolytic therapy is superior to conventional treatment from the viewpoints of medical performance and occurrence of complications [1-4,6-7].

The site of occlusion needs to be taken into account. Sasaki et Al reported that reopening success rates for blood flow might differ considerably, with values of 69% for middle cerebral artery and 78% for the basilar artery, but only 20% for internal carotid artery [7]. In case of acute stroke involving the carotid artery, no improvement of clinical conditions may be observed even though reopening is attained. The incidence of haemorrhagic complications within 24 hours also varies with the occlusion region, with reported rates of 16% for the middle cerebral artery, 22% for the basilar artery, and a high of 40% for the internal carotid artery. The outcome of patients with ICA occlusion thus is worse than with other regions, largely due to the amount of thromboembolus, requiring a long period of medical treatment with high dose of fibrinolytic agents, resulting in enlargement of cerebral infarction and increasing haemorrhagic complications [9]. Also, ICA thrombi may migrate into peripheral regions with consequent increase in morbidity.

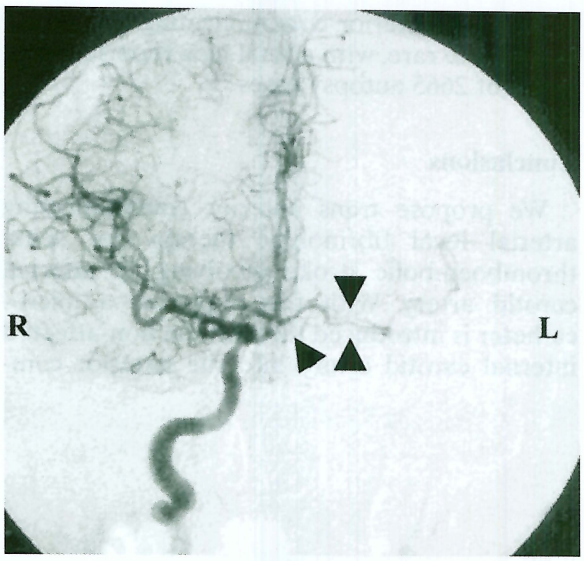

Figure 6 Right internal carotid arteriogram, 7 days after thrombolytic therapy, AP view, showing left A1 and M1 proximal througn anterior communicating artery (arrow).

Our technique proposed here is a trans anterior communicating arterial local fibrinolytic therapy. The distinction is to introduce a micro catheter by way of the intact internal carotid artery through the anterior communicating artery and anterior cerebral artery without advance of catheter into the thromboembolus in the internal carotid artery at the affected site. This technique makes it possible to decrease the time for treatment and to decrease the dose of therapeutic agents. In the two cases introduced here, the dose of urokinase was 600,000 units and the time needed for treatment was three to five hours. Reopening of blood flow was attained within a shorter time than normally necessary. The proposed technique did not cause haemorrhagic complications and the clinical outcome was very satisfactory in both of our cases.

The possibility of anatomical restrictive conditions because of use of the collateral circulation must be considered. From anatomical examination of the anterior communicating artery and A1, indispensable for our technique, the diameter of anterior communicating artery is 0.67~2.11 mm (average 1.36 mm), A1 (right) 1.17~2.34 mm (average 1.76 mm) and A1 (left) 1.33~2.44 mm (average 1.89 mm) [10].

This suggests that there should be no problem with introduction of the size of microcatheter employed here. In the literature the

absence of anterior communicating artery and A1 is quite rare, with a total lack reported for a series of 2665 autopsy cases [11].

Conclusions

We propose trans anterior communicating arterial local fibrinolytic therapy for acute thromboembolic stroke involving an internal carotid artery. With this approach a microcatheter is introduced through the non-affected internal carotid artery into the anterior communicating artery and advanced via the A1 and M1 to the affected site. This method has mayor advantages in allowing a decrease in the dose of fibrinolytic agent and the time required for the endovascular technique. Migration of the thrombus located in the affected ICA can also be avoided and it is expected to have satisfactory clinical effectiveness.

This technique for acute thromboembolic stroke involving the internal carotid artery should be considered when difficulties could arise with conventional treatment approaches.

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